

CONNECTING via Winsock to STN

Trying 31.61.91...open

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RELATION PATHOS IN FILE 'MAILING' AT 19:17, EMBASSY, S. AMERICA
AT 19:17, 19 AUG 1991.
FILE 'MAILING' ENTERED AT 19:17, 19 AUG 1991.
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FILE 'BIOESIS' ENTERED AT 19:17, 19 AUG 1991.
COPYRIGHT © 1991 BIOESIS P
STN INTERNATIONAL LOGOFF AT 19:17:41 ON 19 AUG 1991.

CONNECTING via Winsock to STN

Trying 31.61.91...open

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TERMINAL ENTRY 1, 2, 3, 4, 5, 6, 7, 8

* * * * * Welcome to STN International! * * * * *
NEWS 1 Web Page URLs for STN Seminar Schedule - N. America
NEWS 2 Iss 1 The CA Lexicon available in the CAUC and A files
NEWS 3 Feb 6 Engineering Information Encapsus files have new names
NEWS 4 Feb 16 TOALINE no longer being updated
NEWS 5 Apr 21 Search termset WIPILEX by chemical structure
NEWS 6 Apr 21 PBR-1997 REFERENCES NOW SEARCHABLE IN CAUC AND CA
NEWS 7 May 12 LIGENE Release
NEWS 8 Jun 1 Published patent applications AI are now in WIPILEX
NEWS 9 Jul 1 New Site alert frequency now available in Internet's
TWI and DII

NEWS EXPERTS: Allstet is CURRENT WIND WS VERSION IS V1.0,
USWIND MADING B VERSION IS V1.0, ENW AND P.1.0,
AND CURRENT CINCLIVE FILE IS VATH 1.0 AUGUST 1991
NEWS HOURS: STN operating hours plus Help Desk Availability
NEWS INTER: General Internet Information
NEWS LOGIN: Welcome Banner and News Items
NEWS PHONE: Direct Dial and Telecommunication Network Access to STN
NEWS WWW: CAS World Wide Web Site general information.

Enter NEWS followed by the item number or name to see news on that specific topic.

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* * * * * STN - Topics * * * * *

FILE 'THIMBY' ENTERED AT 19:17:41 ON 19 AUG 1991

File file medline_cplus_errmase_rislist
USD IN U.S. DOLLARS
SINCE FILE TOTAL
FILE ESTIMATE: 1.01
ENTRY 1.01
VERSION 1.01

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FILE 'BIOESIS' ENTERED AT 19:17:41 ON 19 AUG 1991.

* * Antibody INN Interferon gamma or IFN gamma or gamma interferon or interferon
IFN gamma antibody INN INTERFERON GAMMA or IFN GAMMA or GAMMA
INTERFERON OR GAMMA IFN

* * Antibody INN TNF or TNF alpha or tumor necrosis factor alpha or TNF beta or tumor necrosis factor beta
TNF alpha antibody INN TNF or TNF ALPHA or TUMOR NECROSIS FACTOR
ALPHA or TNF BETA or TUMOR NECROSIS FACTOR BETA

* * Antibody INN TNF alpha or tumor necrosis factor alpha
TNF alpha antibody INN TNF ALPHA or TUMOR NECROSIS FACTOR ALPHA

* * Antibody INN TNF beta or tumor necrosis factor beta

* * Antibody INN TNF gamma or gamma interferon

* * Antibody INN TNF necrosis factor or tumor necrosis factor

* * Antibody INN TNF necrosis factor alpha

* * Antibody INN TNF necrosis factor beta

* * Antibody INN TNF necrosis factor gamma

* * Antibody INN TNF necrosis factor necrosis factor

* * Antibody INN TNF necrosis factor necrosis factor alpha

* * Antibody INN TNF necrosis factor necrosis factor beta

* * Antibody INN TNF necrosis factor necrosis factor gamma

111 APP REM 11
PROCESSING COMPLETED FOR LS
111 63 SUR REM 38 113 DUPLICATES REMOVED

100 I will say AIDS is acquired immunodeficiency disease
101 I will say AIDS is AQUED IMMUN DEFICIENT DISEASE

$\Rightarrow \text{P}(\text{A}) = \frac{\text{Number of favorable outcomes}}{\text{Total number of outcomes}} = \frac{1}{6}$

ALL ANSWER IS FOLLOWS
A TENSION NUMBER: MEDLINE
C E M E N T N U M B E R : 00144-47 MEDLINE
T I T L E : PLMNet 101-10544
Last record six part 7, all new in the association with other, but still IFN-pmra, as was stated with first page number 101, 7, 10544, and 10545.
A U T H . P : Bernaghi L B, et al.
P R E P A R A T O R P : PLMNet Institute of Radiology and Infectious Diseases,
M. S. I. Research Institute of Radiology, University of Milan, Italy.
U N I T A R Y N U M B E R : 101-4114 ITALY
S U P E R : JOURNAL OF INFECTION, 1990 May, 14, 1, 1-11.
JOURNAL CODE (ISB): 0893-9039, ISSN: 0893-9039.
P U B. C O U N T R Y : United States
L A N G U A G E : Journal Article; JOURNAL ARTICLE.
E N G L I S H : English
A B R I D G E D I N D E X M E D I C U S J O U R N A L S ; P R I O R I T Y J O U R N A L S
1990's
Entered STN: 19900308
Last Updated or STN: 19970103
Entered Medline: 19980603

Organisms belonging to the *Mycobacterium avium* complex (MAC) are the most common bacterial pathogens in patients with AIDS but factors associated with the activation of cellular defense mechanisms against this atypical mycobacterium have not been defined. Peritoneal macrophages harvested from a chronic MAC infection in C57 black mice are able to kill approximately 6-7% of intracellular MAC. In contrast to T cell killing by unstimulated human and mouse macrophages in vitro, the availability of tumor TNF-alpha, IL-1 β , or GM-CSF permitted evaluation of the role of each of these lymphokines in mice, alone or in combination, in activating macrophages in vitro to kill MAC. Human macrophage-derived macrophages were cultured in vitro, stimulated with IL-1 β , IFN-gamma, or TNF-alpha and infected with MAC (serovars I and II). Mouse peritoneal macrophages were harvested, cultured in vitro, and stimulated with IFN-gamma, TNF-alpha, or GM-CSF. When administered at 24 or 48 h after macrophage infection or when administered 48 h after consecutive days after infection, TNF-alpha (10 ng/ml), both human and murine IFN-gamma were associated with increased intracellular killing of MAC (~10-14% for the mouse and ~1-3% for human macrophages). However, intracellular killing of MAC was not increased with GM-CSF when administered after 48 h post-infection with IFN-gamma. This latter effect was found to be partly anti-TNF antibody, which inhibited the active TNF component in the intracellular killing of MAC. Human macrophages stimulated with GM-CSF without IFN-gamma exhibited increased intracellular killing of MAC, but this increase was not significant. In contrast, when GM-CSF was present, but subsequent MAC killing was measured 48 h after TNF-alpha treatment, the subsequent MAC killing was significantly increased with TNF-alpha. Treatment of macrophages with GM-CSF and TNF followed by IL-1 β (GM-CSF/TNF/IL-1 β) was associated with 66% of intracellular killing. TNF seems to be an important molecule, promoting activation of mycobactericidal mechanisms in human macrophages.

mycobacterium mechanisms in human macrophages. Organisms belonging to the *Mycobacterium avium* complex ('MAC') are the most common bacterial pathogens in patients with AIDS but factors associated with the activation of cellular defense mechanisms against this atypical mycobacterium have not been defined. Fertilization macrophages, of intracellular killing of MAC $\text{S} = 1 - 5$, even when utilized 24 : 48 h after macrophage infection or when administered for 5 consecutive days after infection $\text{S} = 1 - 5$. Both human and murine IFN-gamma were associated with increased intracellular growth, $\text{S} = 1 - 5$, for human macrophages. However, intracellular killing $\text{S} = 1 - 5$ compared with control, was observed after 1 day of treatment with IFN-gamma. This latter effect was fully blocked by anti-IFN antibody, whereas little, if any, effect was observed with the intracellular killing of MAC by human T cells. In fact, IFN plus either IFN-gamma or IL-12,

ANSWER TO PRACTICE EXERCISES

AMERICAN NUMBER:
DOCUMENT NUMBER:
TITLE:

1941-10-10-10000
AM-10-10-10000
AM-10-10-10000

AIDS
Ward, D.: AIDS and the Child, *Pediatrics*, 1985, 75, 101-105.

RE: STATE OF MICHIGAN
DEPARTMENT OF STATE
AGENCY OF RECORDS
TREASURER'S OFFICE
AND THE STATE OF MICHIGAN

ANSWER: The answer is 1000. The total number of students in the school is 1000.

MP The effects were measured 1 hr. of administration of anti-tumor necrosis- α -4 IgG on the survival rate and tumor regression. TNF- α and TNF- β stimulate the LPS-M cell virus infection of female C3H/He mice. The present experiments examined the tumor suppression and cytotoxicity of splenic macrophages. NK cell activity, T- and B-cell proliferation, and T- and T_H cell activity, and the cytotoxicity of LPS, TNF- α , TNF- β , and IgG, and anti-tumor necrosis factor- α IgG, and anti- α -4 IgG, and tumor regression factor- β IgG. These factors were used to test, as they are usually altered dramatically after murine retrovirus infection. Administration of TNF- α IgG and anti- α -4 IgG significantly prevented both virus-induced suppression of splenic NK cell activity, and splenic T and B cell proliferation. They also significantly reduced both virus-induced tumor growth in mice. IgG- α -4 IgG had no effect on the cytotoxicity of LPS and TNF- β IgG against LPS-M cell virus infection. They presented the results of the effect of IgG- α -4 IgG on the cytotoxicity of LPS, TNF- α , TNF- β , and IgG, and anti- α -4 IgG, and tumor regression factor- β IgG.

and elevated splen weight and hyperplastic lymphoma, previous signs of severe immunodeficiency seen during the course of AIDS. These findings suggest that the roles of immunosuppression in AIDS treatment as well as the mechanisms by which retrovirus infection initiates T-cell dysregulation, facilitating immunodeficiencies in AIDS.

II Anti-IL-4 mice, adult type, anti-IFN-gamma, murine, treatment of murine lymphoproliferation induced by murine retrovirus infection during murine AIDS

AB This study was to determine whether administration of anti-interleukin-4 monoclonal antibody (Ab), anti-interferon-gamma (IFN-gamma), and their combination after LPS-M cell-free virus infection of female C57BL/6 mice will prevent their virus-induced suppression of immunosuppression and cytokine dysregulation. Spleen natural killer (NK) cell activity, T- and B-cell proliferation, and T-helper type 1 (Th1) and Th2 cytokine (IL-1, IFN-gamma, IL-6 and IL-10) secretion were monitored, as they are usually altered dramatically after murine retrovirus infection. Administration of IFN-gamma and anti-IL-4 significantly prevented retrovirus-induced suppression of splenic NK cell activity, and splenic T- and B-cell proliferation. They also significantly slowed retrovirus-induced elevation of Th1 cytokine (IL-6 and IL-1) release and monocyte IL-6 and TNF-alpha secretion by splenocytes. They prevented the loss of Th1 cytokine (IL-6 and IFN-gamma) release by spleen cells, and alleviated spleen weight and hyperplasia, lymphoma, peritoneal lymphoma development of acquired immunodeficiency, similar to AIDS. These findings elucidate mechanisms of the course of murine retrovirus infection and the mechanisms by which retrovirus infection induces AIDS.

ST AIDS murine cell culture by interleukin-4 interferon-gamma

II Anticancer

PL: BAC Biological activity or effector, except adverse; B10L Biological study

anti-interleukin-4 monoclonal antibody and IFN-gamma, retardation of immune dysfunction and cytokine dysregulation during murine AIDS

II Lymphokines and Cytokines

RL: ALV Adverse effect, including toxicity; MFM Metabolic formation; B10L Biologival study; FORM Formation, nonpreparative cytokine interleukin-4 and IFN-gamma, administration in murine AIDS effect on formation of

II Spleen, increase fibroplasia, anti-interleukin-4 monoclonal antibody and IFN-gamma, administration in murine AIDS effect on

II Lymphokines and Cytokines

PL: BAC Biological activity or effector, except adverse; B10L Biological study; interleukin-4, anti-interleukin-4 monoclonal antibody and IFN-gamma, retardation of immune dysfunction and cytokine dysregulation during murine AIDS

II Lymphokines and Cytokines

RL: ALV Adverse effect, including toxicity; MFM Metabolic formation; B10L Biologival study; FORM Formation, nonpreparative interleukin-5, anti-interleukin-4 monoclonal antibody and IFN-gamma, administration in murine AIDS effect on formation of

II Lymphokines and Cytokines

PL: BAC Biologival study; FORM Formation, nonpreparative interleukin-5, anti-interleukin-4 monoclonal antibody and IFN-gamma, administration in murine AIDS effect on formation of

II Lymphokines and Cytokines

RL: ALV Adverse effect, including toxicity; MFM Metabolic formation; B10L Biologival study; FORM Formation, nonpreparative tumor necrosis factor-alpha, anti-interleukin-4 monoclonal antibody and IFN-gamma, administration in murine AIDS effect on formation of

II Interferons

PL: BAC Biological activity or effector, except adverse; B10L Biological study; gamma, anti-interleukin-4 monoclonal antibody and IFN-gamma, retardation of immune dysfunction and cytokine dysregulation during murine AIDS

II Interferons, type I, type II, type III

PL: ALV Adverse effect, including toxicity; MFM Metabolic formation; B10L Biologival study; FORM Formation, nonpreparative gamma, retaradation of immune dysfunction and cytokine dysregulation during murine AIDS effect on

** DRS. H.S.

FILE 'HOME' ENTERED AT 16:02:48 ON 19 AUG 2001

FILE 'MEILLINE, CARLOS, EMBACE, RICOS' ENTERED AT 16:03:06 ON 19 AUG 2001
1469 S ANTIBODI ALIN 1 INTERFERON GAMMA OR IFN GAMMA OR GAMMA
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9337 S ANTIBODI ALIN 1 TNF ALPHA OR TUMOR NEPHROSIS FACTOR ALPHA
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Address: 100-10, ADA
Reg. No.: 1994-000000004-
CEN: 10000000000000000000000000000000

1. "MAIN TYPE": Clinical

2. "LANGUAGE": English

AB This study was designed to test, if administration of anti-interleukin-4 (IL-4) monoclonal antibody, rMA, interferon-gamma (IFN-gamma), and their combination after LPS+MLV retrovirus infection of female C57BL/6 mice would prevent retrovirus-induced immunosuppression and cytokine dysregulation. Splenic natural killer (NK) cell activity, T- and B-cell proliferation, anti-T-helper type I (Th1) and Th2 cytokine IL-2, IFN-gamma, IL-1 and TNF-alpha and monokine IL-1beta and tumor necrosis factor-alpha (TNF-alpha), in sera, were monitored, as they are usually altered primarily after murine retrovirus infection. Administration of IFN-gamma and anti-IL-4 significantly prevented retrovirus-induced suppression of spleen NK cell activity, and splenic T- and B-cell proliferation. They also significantly slowed retrovirus-induced elevation of Th2 cytokine IL-2 and IL-10 release and monokine IL-1 and TNF-alpha secreted by splenocytes. They prevented the loss of Th1 cytokine IL-2 and IFN-gamma release by splenocytes, and alleviated splenomegaly and hypergammaglobulinemia, precursors signs of development of acquired immune deficiency syndrome (AIDS). These findings could provide insight into the roles of immunosuppression in AIDS treatment as well as the mechanisms by which retrovirus infection induces cytokine dysregulation, facilitating immunodeficiencies in AIDS.

T1 Anti-IL-4 monoclonal antibody and IFN-gamma administration retards development of immune dysfunction and cytokine dysregulation during murine AIDS

AB This study was designed to test, if administration of anti-interleukin-4 (IL-4) monoclonal antibody (rMA), interferon-gamma, (IFN-gamma), and their combination after LPS+MLV retrovirus infection of female C57BL/6 mice would prevent retrovirus-induced immunosuppression and cytokine dysregulation. Splenic natural killer (NK) cell activity, T- and B-cell proliferation, anti-T-helper type I (Th1) and Th2 cytokine IL-2, IFN-gamma, IL-1 and TNF-alpha and monokine IL-1beta and tumor necrosis factor-alpha (TNF-alpha), in sera, were monitored, as they are usually altered primarily after murine retrovirus infection. Administration of IFN-gamma and anti-IL-4 significantly prevented retrovirus-induced suppression of spleen NK cell activity, and splenic T- and B-cell proliferation. They also significantly slowed retrovirus-induced elevation of Th2 cytokine IL-2 and IL-10 release and monokine IL-1 and TNF-alpha secreted by splenocytes. They prevented the loss of Th1 cytokine IL-2 and IFN-gamma release by splenocytes, and alleviated splenomegaly and hypergammaglobulinemia, precursors signs of development of acquired immune deficiency syndrome (AIDS).

These findings could provide insight into the roles of immunosuppression in AIDS treatment as well as the mechanisms by which retrovirus infection induces cytokine dysregulation, facilitating immunodeficiencies in AIDS.

T2 AIDS and clinical studies, interferon and interleukin

2. "MAIN TYPE": Clinical

2.1 BA Biological activity or effect, except adverse; PI

PI: Biological study, anti-interleukin-4 monoclonal antibody and IFN-gamma, retardation of immune dysfunction and cytokine dysregulation during murine AIDS

2.2 Lymphokines and Cytokines

PI: ADV Adverse effect, including toxicity; MFM Metabolic formation; PI: BA Biological study; FORM Formation, nonpreparative; cytokine formation-inhibiting factor, anti-interleukin 4 monoclonal antibody and IFN-gamma, administration in murine AIDS effect on formation of;

2.3 Spleen, disease
hyperplasia, anti-interleukin 4 monoclonal antibody and IFN-gamma, administration in murine AIDS effect on

2.4 Lymphokines and Cytokines

PI: BA Biological activity or effector, except adverse; PI

PI: Biological study, interleukin 4, anti-interleukin 4 monoclonal antibody and IFN-gamma, retardation of immune dysfunction and cytokine dysregulation during murine AIDS

2.5 Lymphokines and Cytokines

PI: AD Adverse effect, including toxicity; MFM Metabolic formation; PI: BA Biological study; FORM Formation, nonpreparative; interleukin 4, anti-interleukin 4 monoclonal antibody and IFN-gamma, administration in murine AIDS effect on formation of

2.6 Lymphokines and Cytokines

PI: AD Adverse effect, including toxicity; MFM Metabolic formation; PI: BA Biological study; FORM Formation, nonpreparative; interleukin 4, anti-interleukin 4 monoclonal antibody and IFN-gamma, administration in murine AIDS effect on formation of tumor necrosis factor alpha, anti-interleukin 4, anti-interleukin 4 antibody and IFN-gamma, administration in murine AIDS

2.7 Interferons

PI: BA Biological activity, r effect t, except adverse; PI

PI: Biological study, gamma, anti-interleukin 4 monoclonal antibody and IFN-gamma, retardation of immune dysfunction and cytokine dysregulation during murine AIDS

2.8 Cytokines, PI: Clinical studies

PI: AIV Adverse effect, including toxicity; PI: BA Biological study, gamma, retardation of immune dysfunction and cytokine dysregulation, anti-interleukin 4, anti-interleukin 4 and IFN-gamma, administration in murine AIDS effect on

2.9 Adverse effect, including toxicity; PI: BA Biological study, gamma, anti-interleukin 4, anti-interleukin 4 and IFN-gamma, administration in murine AIDS effect on

2.10 PI: AIV Adverse effect, including toxicity; PI: BA Biological study, gamma, anti-interleukin 4, anti-interleukin 4 and IFN-gamma, administration in murine AIDS effect on

2.11 PI: BA

2.12 PI: Clinical studies

2.13 PI: Clinical studies, PI: Clinical studies, PI: Clinical studies

2.14 PI: Clinical studies, PI: Clinical studies, PI: Clinical studies

114 ANSWER F-1
ASSIGNMENT NUMBER:
DOCUMENT NUMBER:
TITLE:
INVENTOR F-1:
PATENT ASSIGNEE S-1:
COURT(S):
DOCUMENT TYPE:
CLASSIFICATION:
FAMILY AND NUMBER:
PARENTING INFORMATION:

APRIL 1, 1997 U.S.A. (EXCLUSIVE)
144611-171001
19-11111
Treatment of autoimmune diseases, including
AIDS
Skurkovich, Boris; Skurkovich, Simon
V.
Advanced Biotherapy Concepts, Inc., USA
U.S. 1, 111, 111, 111-in-part of U.S. 5, 6, 111, 111,
111, 111, 111, 111
Patent
English

AB The present disclosure concerns the treatment of a patient with autoimmune disease, including AIDS, by neutralizing, removing or inhibiting different types of interferons, tumor necrosis factor, HLA class II antigens, IgE, and other pathogenic factors and/or their receptors, as well as neutralizing, removing or inhibiting autoantibodies, including antibodies to target cells, CD4 cells and DNA. Treatment comprises administration of an autoimmune inhibitor, or extracorporeal exposure of the patient's fluid to an immunosorbent comprising an autoimmune inhibitor, followed by return of the treated fluid to the patient, or it comprises a combined therapy involving extracorporeal immunosorption in conjunction with the administration of an autoimmune inhibitor. Combination of a plurality of two or more antisera selected from anti-alpha interferon receptor, anti-beta interferon receptor, anti-gamma interferon receptor, anti-tumor necrosis factor receptor, and anti-interleukin-2 receptor. These are also set.

REFERENCE COUNT: 28
 REFERENCES :
 1 Aetomar Arthritis Rheum 1991, Vol 34, No 1, Pg 1-11
 MEDLINE
 2 Allin W. Radiology 1991, REPRINT
 3 Bannerman J. Radiology 1991, Vol 181, Pg 101-102
 4 Bransford J. Radiology 1991, Vol 181, Pg 103-104
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 8 Carpenter J. Radiology 1991, Vol 181, Pg 111-112
 9 Carpenter J. Radiology 1991, Vol 181, Pg 113-114
 10 Carpenter J. Radiology 1991, Vol 181, Pg 115-116
 11 Carpenter J. Radiology 1991, Vol 181, Pg 117-118
 12 Carpenter J. Radiology 1991, Vol 181, Pg 119-120
 13 Carpenter J. Radiology 1991, Vol 181, Pg 121-122
 14 Carpenter J. Radiology 1991, Vol 181, Pg 123-124
 15 Carpenter J. Radiology 1991, Vol 181, Pg 125-126
 16 Carpenter J. Radiology 1991, Vol 181, Pg 127-128
 17 Carpenter J. Radiology 1991, Vol 181, Pg 129-130
 18 Carpenter J. Radiology 1991, Vol 181, Pg 131-132
 19 Carpenter J. Radiology 1991, Vol 181, Pg 133-134
 20 Carpenter J. Radiology 1991, Vol 181, Pg 135-136
 21 Carpenter J. Radiology 1991, Vol 181, Pg 137-138
 22 Carpenter J. Radiology 1991, Vol 181, Pg 139-140
 23 Carpenter J. Radiology 1991, Vol 181, Pg 141-142
 24 Carpenter J. Radiology 1991, Vol 181, Pg 143-144
 25 Carpenter J. Radiology 1991, Vol 181, Pg 145-146
 26 Carpenter J. Radiology 1991, Vol 181, Pg 147-148
 27 Carpenter J. Radiology 1991, Vol 181, Pg 149-150
 28 Carpenter J. Radiology 1991, Vol 181, Pg 151-152

Skurkovich, Boris; Skurkovich, Simon V.
The present disclosure concerns the treatment of a patient with an immune disease, including AIDS, by neutralizing, removing or inhibiting different types of interferon, tumor necrosis factor, HLA class II antigens, IgE, and other pathogenic factors and/or their receptors, as well as neutralizing, removing or inhibiting autoantibodies, including antibodies to target cells, CD4 cells and TNA. Treatment comprises administration of an autoimmune inhibitor, or extracorporeal exposure of the patient's fluid to an immunosorbent comprising an autoimmune inhibitor, followed by return of the treated fluid to the patient, or it comprises a combined therapy involving extracorporeal immunosorption in conjunction with the administration of an autoimmune inhibitor. Combination of a plurality of two or more components selected from anti-alpha interferon receptor, anti-beta interferon receptor, anti-gamma interferon receptor, anti-tumor necrosis factor or receptor, and anti-interleukin-2 antibodies are disclosed.

receptors), and anti-angiogenesis antibodies are discussed.

AIDS disease

Antikinetics synapsitidis

Autoimmune disease

Cannabis syndrome

Central nervous system

Central nervous system

Chronic fatigue syndrome

Diabetes mellitus

Insulin dependent diabetes mellitus

Demyelination

Multiple sclerosis

Pathogen

Pellagra

Phenylketonuria

Phenylketonuria

Postural orthostatic tachycardia syndrome

Posture

Transplant rejection

Transplant rejection

AIDS

(1) Antiretroviral
 PII-BPN Bi-Symmetric Preparation; TNF Receptor-Use; PI
 Bi-Symmetric Preparation; TNF Use
 interferon-antiviral; interferon-antiviral is anti-extravascular
 TNF; a protein for treatment of autoimmune diseases including
AIDS

(2) Class of HIV antigen

(3) PII-BPN Bi-Symmetric Preparation; Bi-Symmetric
 preparation, which has low antigenicity and extra TNF
 receptor, used for treatment of autoimmune diseases including
AIDS

(4) ANSWER 1 OF 1: ANSWER: DIVERGENT; 1 AY

4. EDITION NUMBER: 1993-1994 AIDS

5. EDITION NUMBER: 1993-1994 AIDS

TITLE: Treatment of autoimmune diseases, including
AIDS, by removal of interferons, TNF and
 TNF receptor

AUTHOR(S): Skurkovich, Simon V.; Skurkovich,
 Boris

PATENT ASSIGNMENT: Advanced Bi-therapy, Inc., USA

CITY: City, USA

COUNTRY: USA

PATENT TYPE: Patent

LAW: English

FAMILY ACC. NUM. COUNT: 3

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 5064443	A	1997-06-06	US 1993-25436	1993-02-09
US 5494111	A	1996-03-06	US 1996-771831	1996-12-03
US 5601261	AI	1996-02-06	US 1994-795783	1994-02-28
PATENT INFORMATION:			US 1993-25436	AI 1993-02-09
			US 1996-771831	AI 1996-12-03
			US 1997-5995783	AI 1997-02-28

AB: The present disclosure concerns a treatment for autoimmune diseases, including **AIDS**, by removing interferons, TNFs and receptors thereof, from body fluids. An extracorporeal real device expels fluids from a patient, including blood, plasma, cerebrospinal fluid, etc., to an immunosorbent to accomplish removal. Following treatment, the fluid is returned to its source. A diagram showing an extracorporeal device for removing pathogenic substances from joint and spinal fluids is included.

TL: Treatment of autoimmune diseases, including **AIDS**, by removal of interferons, TNFs and their receptors

IN: Skurkovich, Simon V.; Skurkovich, Boris

AB: The present disclosure concerns a treatment for autoimmune diseases, including **AIDS**, by removing interferons, TNFs and receptors thereof, from body fluids. An extracorporeal real device expels fluids from a patient, including blood, plasma, cerebrospinal fluid, etc., to an immunosorbent to accomplish removal. Following treatment, the fluid is returned to its source. A diagram showing an extracorporeal device for removing pathogenic substances from joint and spinal fluids is included.

TL: Treatment of autoimmune diseases, including **AIDS**, by removal of interferons, TNFs and receptors thereof, removal with an immunosorbent for treatment of autoimmune diseases, including **AIDS**

(5) AIDS disease

Asetic fluid

Autoimmune diseases

Body fluid

Cerebrospinal fluid

Extracorporeal circulation

Joint (anatomical)

Plasma (blood)

Synovial fluid

 interferon, TNF, and receptor removal with extracorporeal immunoadsorbent for treatment of autoimmune diseases, including **AIDS**

AIDS

Antibodies

Monoclonal antibodies

PII-BPN Treatment-Use; TNF; Bi-Symmetric; PI; PII-BPN
 Treatment-Use; TNF; Bi-Symmetric; PI
 interferon, TNF, and receptor removal with extracorporeal
 immunoadsorbent for treatment of autoimmune diseases, including
AIDS

(6) Antigens

Interferon receptors

Interferon alpha

Interferon alpha receptors

Interferon beta

Interferon gamma

Interferon gamma receptors

Interferons

 removal with extracorporeal

 immunoadsorbent for treatment of autoimmune diseases, including
AIDS

AIDS

Beta-lymphocyte

 plasma proteins; interferon, TNF, and receptor removal with
 extracorporeal immunoadsorbent for treatment of autoimmune diseases,
 including **AIDS**

(7) ANSWER 4 OF 1: MEDLINE

 (COPYRIGHT)

APPLICATION NUMBER: PII-BPN-4- MEDLINE

Edition NUMBER: PII-BPN-4- I ALMed III -#74036

TITLE: Removal and characterization of the interferon-alpha
 present in patients with advanced human immunodeficiency
 virus type I disease.

AUTHOR(S): Skurkovich, Boris; Skurkovich, Boris-Peter; Skurkovich, Boris
 Skurkovich, Boris-Peter; Skurkovich, Boris-Peter; Skurkovich, Boris-Peter
 Skurkovich, Boris-Peter; Skurkovich, Boris-Peter; Skurkovich, Boris-Peter

AB: AIDS; TNF; Bi-Symmetric; PI; PII-BPN; Treatment-Use; TNF; Bi-Symmetric; PI

NLA: 1993-02-09; AIDS
 Bi-Symmetric; PI; PII-BPN; Treatment-Use; TNF; Bi-Symmetric; PI

SEARCHED
INDEXED
SERIALIZED
FILED
APR 19 1987
FEDERAL BUREAU OF INVESTIGATION - WASHINGTON
U.S. DEPARTMENT OF JUSTICE

LAST NAME: HALL, JR.
FIRST NAME: RONALD
MIDDLE NAME: L
SEX: MALE
DATE OF BIRTH: 1941
ENTRY DATE: Entered FBI Lexis 1
Last Updated 1 JUN 1993
Entered Medline 1993-06-03

AB To examine a possible association between plasma viremia and interferon-alpha, IFN-alpha, in patients with the acquired immunodeficiency syndrome AIDS, we performed IFN plasma immunoadsorption apheresis in four volunteers with AIDS who had sustained levels of endogenous plasma IFN-alpha. IFN-alpha apheresis with two plasma volume exchanges was performed daily for 5 days. Clinical signs and symptoms and hematologic, virologic, and immunologic parameters were monitored. Two subjects developed anemia from phlebotomy, and one had a catheter-associated arteritis. The IFN-alpha apheresis was effective only in transiently reducing IFN-alpha levels. In IFN-alpha low day it's rapid reduction in cellular virus HIV-1 was observed, but three of four subjects had a persistent increase in cellular plasma virus burden following the procedure. On the contrary, the low IFN-alpha level, apheresis also with its broad antiviral properties, characterization. The HIV-1 IFN-alpha showed characteristics of ELISA, western blot, and indirect assays similar to the substances in the natural protein. The natural recombinant and HIV-associated IFN-alpha's antiviral activity, mainly against anti-HIV, anti-HIV, and in some patients with AIDS affected viral burden likely reflects properties of the virus or host. This fact is independent of IFN-alpha.

AB ... published by Stevens M, L'Angelie P, Au Eddy P, Klein P, Lin M, D'Costa J; Med Sci Rev Bellanti J; Skurkovich S; L'Angelie H F

AB To examine a possible association between plasma viremia and interferon-alpha, IFN-alpha, in patients with the acquired immunodeficiency syndrome AIDS, we performed IFN plasma immunoadsorption apheresis in four volunteers with AIDS who had sustained levels of endogenous plasma IFN-alpha. IFN-alpha apheresis with two plasma volume exchanges was performed daily for 5 days. From the chronic was not acid labile. The inability of large amounts of plasma IFN-alpha to bind to some patients with AIDS affected viral burden likely reflects properties of the virus or of host factors independent of IFN-alpha.

ANSWER 5 OF 10 MEDLINE DUPLICATE 3
ACCESSION NUMBER: 94254741 MEDLINE
DOCUMENT NUMBER: 94254741 PubMed ID: 7515145
TITLE: A disturbance of interferon synthesis with the hyperproduction of unusual kinds of interferon can trigger autoimmune disease and play a pathogenetic role in AIDS: the removal of these interferons can be therapeutic.
AUTHOR: Skurkovich S; Skurkovich B; Bellanti J
PUB DATE/TYPE: Advances Biotherapy Concepts Late, Rockville, MD.
JOURNAL: Medical Biotherapeutics, 1994 Jan; 4(1):1-17. Ref ID: 94254741
JOURNAL: Bellanti J
JOURNAL: United States
JOURNAL: Advances Biotherapy Concepts Late
GENERAL: Review, Review
LAW STATUS: English
FIELD/MENTH: Immunology
ENTRY/Month: 1994
ENTRY DATE: Entered STN: 1-6-94
Last Updated in STN: 1997-07-3
Entered Medline: 1993-06-03

AB Disturbances of interferon synthesis will the hyperproduction of unusual kinds of interferon may be the initial step which triggers all chronic disease and with a third type of viral vector can trigger the disturbances of severe immunological and viral diseases. Interferon disturbance of this kind of viral vector can trigger the onset of autoimmune disease or an underlying chronic autoimmune condition can exacerbate or trigger the disease. healthy people do not have interferon in their blood. This fundamental disturbance of interferon synthesis can result either from a genetic predisposition or from the influence of certain viruses or viral particles or both factors together. AIDS has many features similar to autoimmune disease, including the hyperproduction of aberrant interferon, a type with restricted anti-HIV activity, protectively induced by HIV to allow its continued replication and survival. This interferon stimulates the production of certain cytokines and anti-antibodies which help unleash the potentially self-destructive powers of the immune system, bringing immunological chaos. In other words, while usual viruses induce normal interferon, which protects the cells against viral infection, HIV induces an abnormal, defective kind of interferon which ensures virus survival. Since there is no more effective method of destroying HIV directly, removing lines in this basic function could directly destroy HIV and possibly help treat the immunosuppression. AB 1993-06-03 07:11:56 818

AB ... interferon synthesis will the hyperproduction of unusual kinds of interferon can trigger autoimmune disease and play a pathogenetic role in AIDS: the removal of these interferons can be therapeutic.
AB Skurkovich S; Skurkovich B; Bellanti J
AB ... result either from a genetic predisposition or from the influence of certain viruses or viral particles or both factors together. AIDS has many features similar to autoimmune disease, including the hyperproduction of aberrant interferon, a type with restricted anti-HIV activity, protectively induced by HIV to allow its continued

ANSWER 6 OF 10 MEDLINE DUPLICATE 4
ACCESSION NUMBER: 94254741 MEDLINE
DOCUMENT NUMBER: 94254741 PubMed ID: 7515145
TITLE: A disturbance of interferon synthesis with the hyperproduction of unusual kinds of viral vector can trigger the onset of autoimmune disease or an underlying chronic autoimmune condition can exacerbate or trigger the disease. healthy people do not have interferon in their blood. This fundamental disturbance of interferon synthesis can result either from a genetic predisposition or from the influence of certain viruses or viral particles or both factors together. AIDS has many features similar to autoimmune disease, including the hyperproduction of aberrant interferon, a type with restricted anti-HIV activity, protectively induced by HIV to allow its continued

AB Skurkovich S; Skurkovich B; Bellanti J
AB ...
PUB DATE/TYPE: Advances Biotherapy Concepts Late, Rockville, MD.

ANTI-POLYMER AND IMMUNE DEFICIENCIES
SKURKOVICH S; SKURKOVICH B
FORT WORTH, TX, USA
ASSIGNEE: LIVUS, CAPRAT FIBR, INC
PATENT INFORMATION: US 4-1444212 Apr 1979
TITLE: Off. Gaz. U. S. Pat. Trademark Off., Pat., 1969 11:1 4,
1970 1:1
CLIENT: INVENT. IDN#N: 199-1133.
DOCUMENT TYPE: Patent
FILE SEGMENT: P4V3M1
LAW FIRM: Ehrlich
P1: METHOD FOR TREATING AIDS AND OTHER IMMUNE DEFICIENCIES AND
IMMUNE DISORDERS.
ATT: SKURKOVICH S; SKURKOVICH B

104 ANSWER ID: 1 MEDLINE SUBJECTS: CLOSTRIDIUM
A BACTERIUM NUMBER: 104-001 MEDLINE
L HUMAN NUMBER: 104-001 SUBJECTS: CLOSTRIDIUM
TITLE: A diagnostic test of the immunological type of the
Clostridium system as infected in patients disease in
humans.
AUTHORS: Skurkovich S; Skurkovich B; Belyantsev I

This hypothesis is a continuation of a continuing belief that the interferon IFN system is a cascade of sequentially interacting responses of IFN-alpha, -beta, and -gamma involving amplification of the immune response. We propose that every antigen is an IFNogen. The first stage is of immune responsiveness is associated primarily with the production of the family of IFN-alpha. In certain immunologically mediated diseases, including the autoimmune diseases and AIDS, disturbances in the synthesis of IFN-alpha occur with a switch to the production of predominantly acid-labile types, which have a negative immunoregulatory effect. Moreover, disturbances of IFN synthesis in the embryo or fetus can lead to deformities. Some viruses and other biological and chemical substances manifest a pathologic effect by the IFN they induce. This IFN may help sustain the viruses and other substances which induce this IFN. We think it is unsafe to give patients immunoregulators in incomplete form. Thus, there is a potential danger in giving patients recombinant forms of IFNs and interleukin 3 prepared in bacteria. In certain immune disorders, we may be able to treat patients by the blocking or removal of hyperproduced IFNs from the body. This may result in the restoration of normal IFN balance and normal responsiveness.

AB Skurkovich S; Skurkovich B; Borelli J A
and associated primarily with the production of the family of IFN- α 's in certain immunologically mediated diseases, including the autoimmune diseases and AIDS. Disturbances in the synthesis of IFN- α 's along with a switch to the production of predominantly acidic-type, which have been

ALL INFORMATION CONTAINED AND ANYWHERES HEREIN ARE UNPUBLISHED AT THE REQUEST OF THE SOURCE.

JOHN H. SIBLEY TOTAL
FEDERAL BUDGET 11,144 11,144

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